We are particularly interested in the possible pathophysiologic role of TNF-α in hyperplastic inflammatory diseases such as psoriasis, because we have recently observed numerous dermal dendritic cells which are positive by immunohistochemical staining in active, untreated psoriatic lesions [7].

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EDITOR'S COMMENTS

I am grateful to Dr. Nickoloff for providing the data on which he based the statement that TNF-alpha does not inhibit human keratinocyte proliferation, as requested by Dr. Symington. Since receipt of these papers, a paper by Detmar and Orfanos [1] has addressed in detail the effects of TNF-alpha on keratinocyte proliferation. The evidence in this paper clearly supports the contention that TNF-alpha inhibits keratinocyte proliferation, as also described by Symington [2] and Detmar et al [3].

In the paper by Detmar, 100 U/ml of TNF-alpha provided maximal inhibition of proliferation as judged by both cell number and thymidine uptake. The preliminary data summarized above by Nickoloff seem to indicate that at 100 U/ml of TNF-alpha there was a decrease in keratinocyte number. I read the data, as explained to show that 2.4×10^5 cells increased to 3.4×10^5 over two days in the control, but only to 2.6×10^5 with 100 U/ml TNF-alpha. In fact, compared to the starting point, the inhibition seems impressive.

It is evident that keratinocytes near confluence are not the best system to study inhibition of proliferation. In studies using keratinocytes well before confluence, TNF-alpha has produced significant inhibition of proliferation.

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